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Term: Urokinase-type adj plasminogen and chimeric and liver

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DB Name	Query	Hit Count	Set Name
USPT	Urokinase-type adj plasminogen and chimeric and liver	28	L5
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USPT	Urokinase-type adj plasminogen adj activator and transgenic and liver	15	L3
USPT	Urokinase-type adj plasminogen adj activator and transgenic	21	L2
USPT	Urokinase-type adj plasminogen adj activator	165	L1

09156892

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 2000:210343 CAPLUS
DOCUMENT NUMBER: 132:247157
TITLE: **Urokinase-type plasminogen activator/RAG-2 mouse** with mammalian repopulating hepatocytes for infection by hepadnaviruses
INVENTOR(S): Rogler, Charles E.
PATENT ASSIGNEE(S): Albert Einstein College of Medicine of Yeshiva University, USA
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000017338	A1	20000330	WO 1999-US21838	19990916

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1998-156892 19980918
US 1999-344189 19990624

AB This invention provides a chimeric **mouse** liver model system for mammalian hepatitis. A method is provided for repopulating degenerated liver of immunotolerant mice which lack mature B and T lymphocytes with xenogenic mammalian hepatocytes, particularly primate hepatocytes to generate chimeric mice. In addn., a method of generating a human hepatitis virus-infected chimeric **mouse** is provided. A preferred xenogenic primate hepatocyte is derived from human, chimpanzee, or baboon. These chimeric mice are useful in the investigation of host and viral mechanisms detg. hepadnaviral persistence and hepatocarcinogenesis. Methods for monitoring the development of hepatitis and hepatocellular carcinoma as well as methods for testing and screening anti-viral and anti-cancer compds. with this model system are also provided.

REFERENCE COUNT: 3
REFERENCE(S):
(1) Gupta, S; AM J PHYSIOL PT 1 1999, V277(6), PG1097 CAPLUS
(2) Petersen, J; PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF USA 1998, V95(1), P310 CAPLUS
(3) Ponzetto, A; ITAL J GASTROENTEROL 1991, V23(8), P491 MEDLINE

Q11.N26

L8 ANSWER 10 OF 15 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 95281572 MEDLINE
DOCUMENT NUMBER: 95281572
TITLE: Complete reconstitution of **mouse liver** with xenogeneic hepatocytes.
AUTHOR: Rhim J A; Sandgren E P; Palmiter R D; Brinster R L
CORPORATE SOURCE: Department of Animal Biology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia 19104, USA.
CONTRACT NUMBER: HD-23657 (NICHD)
CA-38635 (NCI)
HD-09172 (NICHD)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1995 May 23) 92 (11) 4942-6.
Journal code: PV3. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199509

QH.N26

AB We have developed a system for studying hepatocellular growth potential in which liver cells are introduced into the diseased livers of albumin-urokinase (Alb-uPA) transgenic mice. To use this system to study xenogeneic cell transplantation, rat liver cells were introduced into immunotolerant Alb-uPA transgenic mice. In regenerated recipient livers, up to 100% of hepatocellular gene expression was of rat origin, demonstrating the creation of a functional mouse liver in which parenchyma is derived from xenogeneic (rat) hepatocytes. Immunotolerant Alb-uPA transgenic mice provide a tool for studying hepatocellular biology of any species, including humans, in a controlled experimental setting.

L8 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 6
ACCESSION NUMBER: 1991:431173 BIOSIS
DOCUMENT NUMBER: BA92:87338
TITLE: COMPLETE HEPATIC REGENERATION AFTER SOMATIC DELETION OF AN ALBUMIN PLASMINOGEN ACTIVATOR TRANSGENE.
AUTHOR(S): SANDGREN E P; PALMITER R D; HECKEL J L; DAUGHERTY C C; BRINSTER R L; DEGEN J L
CORPORATE SOURCE: LAB. REPRODUCTIVE PHYSIOL., SCH. VET. MED., UNIV. PENNSYLVANIA, PA. 19104.
SOURCE: CELL, (1991) 66 (2), 245-256.
CODEN: CELLB5. ISSN: 0092-8674.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB We previously demonstrated that expression of an albumin-**urokinase-type plasminogen activator** (Alb-**uPA**) fusion construct in **transgenic** mice resulted in elevated plasma **uPA** concentration, hypofibrinogenemia, and neonatal hemorrhaging. Two lines of Alb-**uPA** mice were established in which only one half of the **transgenic** pups died at birth; surprisingly, plasma **uPA** concentrations in survivors gradually returned to normal by 2 motnhs of age. The basis for this phenomenon is DNA rearrangement within hepatocytes that affects the transgene tandem array and abolishes transgene expression. Transgene-deficient cells selectively proliferate relative to surrounding **liver**, and this process culminates in replacement of the entire **liver** by clonal hepatic nodules derived from transgene-deficient progenitor cells. In some cases as few as two nodules can reconstitute over 90% of **liver** mass, highlighting the remarkable regenerative capacity of individual **liver** cells.

L6 ANSWER 8 OF 63 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 3
AN 1999:220856 BIOSIS
DN PREV199900220856
TI Zonal regulation of gene expression during liver regeneration of urokinase
transgenic mice.
AU Locaputo, Stephanie; Carrick, Terri L.; Bezerra, Jorge A. (1)
CS (1) Division of Gastroenterology and Nutrition, Children's Hospital Medical Center, 3333 Burnet Ave., Cincinnati, OH, 45229 USA
SO Hepatology, (April, 1999) Vol. 29, No. 4, pp. 1106-1113.
ISSN: 0270-9139.
DT Article
LA English
SL English

DUPLICATE 7

L6 ANSWER 16 OF 63 MEDLINE
AN 1998442697 MEDLINE
DN 98442697
TI Selective repopulation of normal **mouse** liver by Fas/CD95-resistant hepatocytes.
AU Mignon A; Guidotti J E; Mitchell C; Fabre M; Wernet A; De La Coste A; Soubrane O; Gilgenkrantz H; Kahn A
CS INSERM U 129 ICGM, Universite Paris V Rene Descartes, Paris, France.
SO NATURE MEDICINE, (1998 Oct) 4 (10) 1185-8.
Journal code: CG5. ISSN: 1078-8956.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
EW 199901

L6 ANSWER 12 OF 63 BIOSIS COPYRIGHT 2000 BIOSIS
AN 2000:87110 BIOSIS
DN PREV200000087110
TI **Mouse** liver tumorigenesis: Models, mechanisms, and relevance to human disease.
AU Fausto, Nelson (1)
CS (1) Department Pathology, University Washington School Medicine, C-516 Health Sciences Building, Seattle, WA, 98195-7470 USA
SO Seminars in Liver Disease, (1999) Vol. 19, No. 3, pp. 243-252. ISSN: 0272-8087.
DT General Review
LA English

L9 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2001:397008 BIOSIS
DOCUMENT NUMBER: PREV200100397008
TITLE: **Hepatitis** C virus replication in mice with chimeric human livers.
AUTHOR(S): Mercer, David F.; Schiller, Daniel E.; Elliott, John F.; Douglas, Donna N.; Hao, Chunhai; Rinfret, Aline; Addison, William R.; Fischer, Karl P.; Churchill, Thomas A.; Lakey, Jonathan R. T.; Tyrrell, David L. J.; Kneteman, Norman M. (1)
CORPORATE SOURCE: (1) Department of Surgery, Surgical-Medical Research Institute, University of Alberta, Edmonton, AB: nkneteman@cha.ab.ca Canada
SOURCE: Nature Medicine, (August, 2001) Vol. 7, No. 8, pp. 927-933.
print.
ISSN: 1078-8956.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Lack of a small animal model of the human **hepatitis** C virus (HCV) has impeded development of antiviral therapies against this epidemic infection. By transplanting normal human hepatocytes into SCID mice carrying a plasminogen activator transgene (Alb-uPA), we generated mice with chimeric human livers. Homozygosity of Alb-uPA Was associated with significantly higher levels of human **hepatocyte** engraftment, and these mice developed prolonged HCV infections with high viral titers after inoculation with infected human serum. Initial increases in total viral load were up to 1950-fold, with replication confirmed by detection of negative-strand viral RNA in transplanted livers. HCV viral proteins were localized to human **hepatocyte** nodules, and infection was serially passaged through three generations of mice confirming both synthesis and release of infectious viral particles. These chimeric mice represent the first murine model suitable for studying the human **hepatitis** C viru